

# Impact of different polymorphisms of IL 10 in prevalence of liver fibrosis and progression in hepatitis C Patients.

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## Background

HCV induces chronic liver disease, which is characterized by a persistent hepatic parenchyma inflammatory process that may progress to cirrhosis and hepatocarcinoma. Genetic differences among the infected hosts can determine the progression of the infection, causing different individuals to respond in different ways to the viral infection. Cytokines have become the focus of many scientific studies, as there is evidence of the contribution of genetic factors to the imbalance of the inflammatory profile of the patient, which directly affects the clinical outcome and the severity of hepatitis C and other infectious diseases.

## Material/Methods

Our study included data of 244 patients (Average age 39.1 years, 55.7% men, 44.3% female) with chronic hepatitis C (HCV genotype 1 to 4). In addition to many demographic characteristics also laboratory parameters (ALT, AST, g-GT, bilirubin, Quick) and hepatitis-related parameters (hepatitis C viral load, validation of hepatitis A or/and B and duration/type of the anti- HCV therapy) were obtained.

From 186 patients grade of liver fibrosis was determined after METAVIR. Fibrosis abundance and rate of liver fibrosis progression were determined. These parameters were related to chosen genpolymorphism of IL-10 (G/A -1082; C/T -819; -592 C/A), rate of fibrosis progression and prevalence of fibrosis

## Results

The IL-10 G / A -1082 genotypes AA, GA and GG showed no difference in prevalence of liver fibrosis (74.4 to 85.2%), the rate of fibrosis progression and the relative risk fibrosis (1.04 to 1.1 old increases) detectable. At IL-10 C/T -819 the three genotype TT, CT and CC showed no difference in the rate of fibrosis. However, the fibrosis progression in TT was 1.5 fibrosis-classes per year higher than CT (0.9) or CC (1.1). The relative risk for liver fibrosis did not differ (1.09 to 1,15fold).

The IL-10 C/A -592 Genotype CA led the least likely to liver fibrosis (68.5%), while patients with CC (82.5%) and AA genotype (88.2%) more often developed a liver fibrosis.

The AA genotype led to a higher fibrosis progression rate (1.6 fibrosis classes / year) than the CA (0.9 classes) or CC genotype (1.1 classes). However, the relative risk of fibrosis was 0, 93 times lower than the CC genotype compared to AA.

## Conclusions

The IL-10 G / A -1082 genotypes AA, GA and GG showed no difference in prevalence of liver fibrosis, the rate of fibrosis progression and the relative risk fibrosis detectable. At IL-10 C/T -819 the three genotype TT, CT and CC showed no difference in the rate of fibrosis. However, the fibrosis progression in TT was 1.5 fibrosis-classes per year higher than CT or CC. The relative risk for liver fibrosis did not differ.

The IL-10 C/A -592 Genotype CA led least likely to liver fibrosis, while patients with CC and AA genotype more often developed a liver fibrosis. The AA genotype led to a higher fibrosis progression rate . The relative risk of fibrosis was lower than the CC genotype compared to AA.